Chapter 9

NONSMOKERS AND SMOKERS INDOORS

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The recent claim that cigarette smoke is a health hazard to the nonsmoker is based on interpolation of several experimental observations that include elevation of carbon monoxide (greater than 25 ppm) in smoked-filled enclosures such as a sealed chamber or a nonventilated conference room. Actual measurements of carbon monoxide in residential houses, offices and public places show that levels rarely exceed 10 ppm, a concentration that is no different from outdoors.

The epidemiologic studies on children show a relationship of parental smoking to incidence of respiratory symptoms and this correlation has been interpreted as the consequence of exposing children to cigarette smoke. There are other variables in these comparisons which can explain the correlation of parental smoking with respiratory symptoms, such as inherited susceptibility to disease, socioeconomic factors, contagion of infectious diseases, and levels of indoor pollution.

This chapter reviews the literature relating to the effect of cigarette smoke on the nonsmoker. The information is presented according to its source separating measurements made in living quarters and public places from those made in artificial situations created in the laboratory.

A. CONCENTRATION OF CIGARETTE SMOKE INDOORS

The concentration of cigarette smoke indoors is usually estimated by the analysis of carbon monoxide, based on the assumption that its major sources which may become more important than cigarette smoke, such as burners and stoves indoors and vehicular traffic outdoors. The other constituents of cigarette smoke, such as nicotine, acrolein and benzypyrene, have also been used as tracers for cigarette smoke but the techniques are more complex than the methods for analyzing carbon monoxide.

1. Home Environment.

There is no published measurement of carbon monoxide from cigarette smoke in residential houses. To the contrary there are several reports indicating that the carbon monoxide indoors originates from vehicular emissions outdoors, burners for house heating and kitchen stoves.

a. Vehicular traffic outdoors. The carboxyhemoglobin levels of individuals residing in areas of heavy vehicular traffic are higher than those with light traffic. The mean values for residents in Tokyo are as follows:

City	COHb % mean	
	heavy traffic	light traffic
Tokyo	4.27	2.79

The smoking habits of the residents examined by Suzuki et al.[1] in the above results were not known. Oyagi et al.[2] arrived at the same conclusion based on

measurements of carbon monoxide in the expired air. They included that families residing close to traffic absorbed not only carbon monoxide but other motor exhaust gases such as lead and benzypyrene.

Derouane[3,4] compared outdoor and indoor levels of carbon monoxide in private homes in Liege. In the absence of indoor sources of carbon monoxide, the indoor concentration is 66 to 87% of the outdoor level of carbon monoxide.

b. Heating burners. The carboxyhemoglobin levels of residents in Tokyo were compared during the cold months requiring house heating and the months that did not. The results of Watanabe et al.[5] are as follows:

City	COHb Range	
	summer	winter
Tokyo	0.7-2.3	1.5-4.5

The above levels indicate that the use of heaters contributes a significant amount of carboxyhemoglobin. Biersteker and DeGraaf[6] examined homes in Rotterdam during the winter months and reported that the levels of carbon monoxide are

In the United States, Yates[7] examined 1,061 combustion appliances in 372 residential establishments. Forty percent of the homes investigated contained one or more appliances that were found to be emitting carbon monoxide. About 25% of all appliances listed were discharging carbon monoxide into the room atmosphere and 24% of those that were found positive were emitting carbon monoxide in the range of 200 ppm and over. In the house that contained the positive-emitting appliances, 40% of the residents gave clinical histories of mild carbon monoxide exposure.

c. Kitchen burners. The major sources of carbon monoxide in the kitchen are gas burners.[8] and frying pan.[9] Wade et al. sampled the air in four private houses which had gas stoves. The indoor level of carbon monoxide reached as high as three times the outdoor level.

d. Other pollutants indoors. The survey of houses by Wade et al.[10] also revealed that nitrogen oxide and nitrogen dioxide from the concentration of particulates is increased in the daytime and reduced at night when there was no household activity.[11, 12] It is necessary to realize that pollutants other than carbon monoxide are present in the home environment when the suspected diseases associated with cigarette smoke exposure are discussed.

2. Public Places.

The concentrations of carbon monoxide and benzypyrene in public places are influenced by outdoor and indoor sources. The only tracer that is specific for cigarette smoke is nicotine. Carbon monoxide although widely used to estimate levels of cigarette smoke indoors, is not appropriate for this purpose because there are indoor and outdoor sources of carbon monoxide.

a. Office buildings. In an office building in Oslo, von Ubisch and Westerlund[13] continuously monitored the carbon monoxide levels inside a conference room. Although the concentration of carbon monoxide started at 2 ppm in the morning, it rose to 12 ppm in the evening. This increase was not caused by smoking but reflected the denser traffic in the ares which was seen in the concentration of carbon monoxide in the air intake into the building. Godin et al.[14] observed the same phenomenon in an office building in Toronto. The indoor carbon monoxide

concentration mirrored the outdoor level, with a lag of one to two hours. Kimura[15] reported the concentration of offices in Japan ranging from 2 to 11 ppm. In an office building in London, Cole[16] reported the levels of carbon monoxide ranging from 3 to 8 ppm. Harke[17] carried out continous measurements in office buildings located in Hamburg. The mean level in one air conditioned building and in another non-airconditioned building never exceeded 5 ppm. In a room of 78.3 cubic meters occupied by 3 smokers, the 30-min value of 9 ppm was measured and reached a peak of 15.6 ppm.

b. Restaurants and bars. The following levels of carbon monoxide have been reported in the following places:

Vancouver, large cafeteria	[18]	less than 10 ppm
Cincinnati, two taverns	[19]	mean 12.5 ppm

Benzypyrene has also been detected in a large cafeteria and beer parlor in Vancouver,[18] as well as in a restaurant in Prague.[20] However, the kitchen as a primary source of benzypyrene could not be excluded.

c. Theaters, arenas, and stations. The levels reported at public places are as follows:

		CO ppm
Toronto:	theatre auditorium (smoking prohibited)[14]	1.4 ± 0.8
	theatre foyer (smoking permitted)	3.4 ± 0.8

	CO ppm
Vancouver[18]: theatre lobby	less than 10 ppm
political convention	less than 10 ppm
bingo game in large hall	less than 10 ppm

In addition to carbon monoxide, nicotine has been detected in the atmosphere of public places in Hamburg[21] and Boston.[22] The following concentrations were reported by Hinds and First:[22]

Boston (no. of samples)	Nicotine µg/meter^3 (mean)
Bus waiting station (2)	1.0
Airline waiting station (2)	3.1
Cocktail lounge (3)	10.3
Student lounge (1)	2.8
Restaurant (4)	5.2

Hinds and First[22] calculated the equivalent amount of filter cigarettes per hour that would generate the levels of nicotine tabulated above. They concluded that the nonsmoker was inhaling the equivalent of less than one-hundredth of a cigarette per hour.

B. EXPERIMENTAL OBSERVATIONS IN NONVENTILATED ROOMS

The most widely quoted levels of carbon monoxide in smoke-filled rooms were taken from nonventilated or poorly ventilated rooms. The results are as follows:

	no. of cigarettes/ 10 cu m/hour	CO concentration ppm
Harmsen and Effenberger, 1957[21]	6	80
Lawther and Commins, 1970[23]	5	20
Harke et al., 1972[24-28]	3	8
Anderson and Dalhamm, 1973[29]	4	4.5
Russell et al., 1973[30]	20	38

There is no correlation of the number of cigarettes to carbon monoxide concentration because of the variability in ventilation from study to study. The most extreme situation which had a concentration of 80 ppm was in a non-ventilated room. Harke et al.[24-28] compared various rooms at different levels of cigarette consumption. In the same room with fixed numbers of cigarettes, ventilation reduced the level of carbon monoxide from 8 ppm to less than 5 ppm.

1. Carboxyhemoglobin Levels.

Some of the experimental observations listed above included measurements of carboxyhemoglobin levels in nonsmokers before and after exposure in a smoke-filled room.

	CO level	COHb mean ±S.D.(Range) Before	After
Harke, 1970[24]	30	0.9±0.1(0.7-1.1)	2.1±0.1(2.0-2.3)
	5	0.9±0.1(0.7-1.0)	1.3±0.1(1.2-1.6)
Russell et al., 1973[30]	38	1.6±0.6(0.7-2.2)	2.6±0.7(1.7-4.2)

The above results indicate a rise of about 1% COHb at a room concentration of 30 to 38 ppm. In addition, Anderson and Dalhamn[29] reported no change at a carbon monoxide concentration of 5 ppm.

The major shortcoming of the measurements of carboxyhemoglobin levels in subjects and the carbon monoxide concentration in rooms that are poorly ventilated is that there are no control observations to determine diurnal variations in a room without generation of cigarette smoke. It is necessary to subtract the outdoor levels of carbon monoxide from the indoor levels to obtain the amount attributed to cigarette smoke.

2. Nicotine Absorbed.

Harke[24] estimated the amount of nicotine absorbed by the nonsmoker in smoke-filled room to be about 1% of that absorbed by the smoker. In another smoke-filled room, Horning et al. reported the nicotine absorbed by the nonsmoker to be 5% of that absorbed by smokers.[31] The difference between 1% and 5% absorption of nicotine is probably based on the technique for estimation of nicotine absorbed. The 1% amount absorbed by the nonsmoker is close to the less than 1% estimated by Hinds and First based on analysis of air (see A above).

3. Acrolein and Acetaldehyde.

While the amounts of acrolein and acetaldehyde in the ambient air have been analyzed the amounts absorbed have not yet been determined. Harke et al.[27] used

an unventilated room of 38.2 cubic meters filled with smoke generated from cigarettes by a smoking machine; 30 cigarettes were burned within 13 min. The amounts of acrolein and acetaldehyde measured 11 to 23 min later were: 0.45 mg acrolein/ cu m and 6.5 mg acetaldehyde/ cu m. When 5 cigarettes were burned within 13 min, the respective concentrations were 0.07 mg acrolein/ cu m and 1.3 mg acetaldehyde/ cu m. In additional observations using a larger room (170 cu m), the concentrations of acrolein, acetaldehyde and carbon monoxide were as follows:

	acrolein (mg/ cu m)	acetaldehyde (mg/ cu m)	CO ppm
150 cig. in 30 min	0.38	4.2	53
102 cig. in 30 min	0.21	1.9	28
108 cig. in 2 hr.	0.15	1.7	24.5

From the above results, it would appear that there is a direct relationship between the concentrations of acrolein, acetaldehyde and carbon monoxide.

C. EXPERIMENTAL OBSERVATIONS IN CHAMBER FACILITIES

Since the amount of carbon monoxide generated from a burning cigarette can be measured, it has been possible to derive equations on the expected concentration based on the number of cigarettes, size of the chamber and air ventilation.[32-37] The equations have been verified by actual analysis of carbon monoxide in a chamber under controlled conditions.[38,39] and have been used to calculate the necessary ventilation to avoid accumulation of carbon monoxide.[40-44] It has become apparent that the suspended particulate matter from outdoor and indoor sources are not adequately reduced by air conditioning.[45,46] The information derived from chambers are more relevant to spacecraft and submarine than to the residential and public buildings.

D. EFFECTS OF CIGARETTE SMOKE ON THE HEALTH OF NONSMOKERS

The information on the health effects of cigarette smoke is derived in part from the simulated smoke-filled rooms described above (B and C) as well as from surveys and epidemiologic studies. The results have been contradictory

for cardiovascular system, respiratory system and mental performance.

1. Cardiovascular System.

There are only two groups of observations relating to the cardiovascular effects of cigarette smoke on the nonsmoker. The first was made by Luquette et al.[47] who exposed 51 elementary school children in a poorly-ventilated enclosure containing cigarette smoke. An acceleration of heart rate and an increase in systolic and diastolic blood pressure were noted in the course of watching a film. It has not been possible to ascertain if the cardiovascular reaction is caused by the film or the cigarette smoke or both. Harke et al.[26] observed 30 adults in a nonventilated room full of cigarette smoke and found no alterations in the electrocardiogram, blood pressure or pulse frequency in response to the cigarette smoker.

That the two groups of researchers reached distinctly opposite conclusions may be explained by differences in the following factors: (a) Age--children are more likely to react to a social environment than adults who repress their reactions. (b) carbon monoxide concentration in the room--measured by Harke et al.[26] but not by Luquette et al.[47] (c) Ventilation of the room--absent in one study and poor in the other.

2. Incidence of Respiratory Disease in Children.

Cameron[48] has surveyed by telephone 1,000 residences in Denver. The respiratory disease rate for children when members of the family smoked, was between 31 and 33%, as compared to 20 to 29% for children without a smoker in the family. However, the same study failed to find a correlation between smoking and the incidence of disease among adults. The author has explained the paradox as being due to greater anxiety of smokers which makes them more aware of

disease in their children, to the influence of smoking, alcohol and coffee upon progeny in utero, which has weakened resistance for smokers' children; or to the fact that smokers themselves are used to their habit-linked maladies and, therefore, report only the more severe afflictions, unlike nonsmokers. In a subsequent survey of 727 households, Cameron et al.[49-51] found that smokers' children were sick more frequently than those of nonsmokers. While psychological reactions of the children were examined,[52] their allergic background was not explored, so that one cannot exclude this latter factor as a possible cause of the difference in prevalence of illness.

Norman-Taylor[53] examined the records of 5-year old new entrants to school and noted that the history of respiratory troubles grows as the level of domestic smoking increases. The author cautioned against too-hastily excluding such socioeconomic factors and cited as an example of the doubtless compounding of medical and extra-medical influences, the fact that low income families tend to smoke more than high income families.

Colley and his collaborators[54-57] have been surveying children of various age groups. Over the first five years of life, the incidence of pneumonia and bronchitis in the first year of life was associated with parents' smoking habits, i.e. lowest incidence was where both parents were nonsmokers and highest was where both smoked. The association was not consistent after the first year of life to the fifth year. From 6 to 14 years of age, there was a close association between parents' and children's respiratory symptoms independent of parents'

smoking habits. The history of illness in childhood correlates with the incidence of chronic respiratory symptoms in adulthood. The investigators could not exclude a genetic susceptibility of the children to respiratory diseases inherited from their parents. Another factor is the level of pollution from burners indoors[58] and vehicular emissions outdoors,[59-61] both of which influence the incidence of respiratory disease in both parents and children. The association of parental smoking and incidence of children's disease can be explained by these coincidental factors affecting both parents and children.

3. Subjective Reactions of Nonsmokers.

There is a considerable confusion in the nature of the complaints of nonsmokers who object to the presence of cigarette smoke. The most extreme opinion is that it is a syndrome of "severe sensitivity to smoke . . . among the symptoms are loss of memory, light headedness, difficulty in concentration, depressive personality changes, double vision and short blackouts . . . coupled with headaches, eye irritation and nasal congestion."[62] The phenomenon appears to be a psychosocial reaction to the irritant action of tobacco smoke.

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a. Odor and irritation of mucosa. The odors of tobacco smoke derive from the presence of nicotine, pyridine, ammonia, aldehydes, volatile oils and other substances. There is a decrease in odor perception level with an increase in either humidity or temperature.[41] Yaglou[42] investigated nonsmoker complaints to the presence of unpleasant odors as well as to irritation of the eyes, nose or

throat. The strength of tobacco smoke odor was evaluated by the sense of smell alone according to an arbitrary 5 point scale ranging from 0 (to mean imperceptible odor or irritation) and up to 5 (very strong odor or intolerable irritation). A room with 1,410 cubic feet of space was occupied by 3 non-smokers and 6 smokers; the latter consumed approximately 24 cigarettes per hour. There were also observers in an adjoining room who entered the test chamber to appraise the strength of odor and irritation. The test chamber was ventilated with air supplies of 6, 14, 26, 35 and 60 cfm (cu feet/min) per smoker. The ventilation required to remove objectionable cigarette smoke odors was as follows:

observers upon entering the room: no odor when 35 to 40 cfm/smoker per smoker

nonsmokers in the room: 25 cfm/smoker for odor; below 15 cfm for irritation

smokers in the room: unable to smell smoke odor; complained of irritation below 15 cfm/smoker.

On the basis of the above results, Yaglou[42] suggested that ventilation requirements be based on the amount of tobacco consumed in a unit time, rather than on the total number of occupants. Ventilation at a rate less than 15 cfm/smoker caused irritation in both smokers and nonsmokers, whereas perception of odor by the nonsmoker was avoided when the supply of outside air was at least 25 cfm/smoker.

A similar scale for evaluating odor and irritation was used by Halfpenny and Starrett[43] in studies of the ventilation requirements of air carriers. An odor chamber 7 ft long and 103 ft in diameter was constructed to provide space for 8 subjects. The concentration of cigarette smoke in the chamber was defined as the amount of tobacco burned (mg/min) divided by the air flow (cfm). Irritation of the eyes, nose and throat, which was just perceptible at a concentration of 0.6 mg/cu ft, became definite at 1.0 mg/cu ft and was judged objectionable at 3.0 mg/cu ft. Smoking surveys were conducted during 9 flights, sponsored by the Federal Aviation Administration.[63] The data showed a remarkably constant percentage of smokers among the passengers (54.8%). Collection of cigarette butts allowed calculation of the amount of tobacco smoked/smoker/hour, which amounted to 0.807 g with a standard deviation of 0.222. On the basis of statistical data concerning national smoking habits, the authors predicted that the most probable contamination rate aboard commercial aircraft is 0.412 g tobacco consumed/passenger/hour. Thus, the current aircraft practice of supplying 25 cfm/min of fresh air per person is adequate to control odor and irritation from cigarette smoke. Health aspects of smoking in transport aircraft have been reviewed by the Federal Aviation Administration.[63]

Johansson and Ronge[64] used an odor chamber with a space of 6.7 cu m. The ventilation required to avoid eye irritation in the nonsmoker was 12 cu m? hour/cigarette; to prevent nose irritation the requirement was 32; and to eliminate annoying odors in dry and warm air, it was 50.

b. Tobacco Hypersensitivity Versus Irritation. There is some confusion in identifying the effects of cigarette smoke on nonsmokers, whether it is a form of allergy or a form of irritation, as described above. For the bronchial asthmatic, the earliest report of irritation to cigarette smoke was made by Hogner in 1920. He described a child suffering from bronchial asthma which he attributed to smoking by members of his family.[65] Brown has estimated that 1% of asthmatics are allergic to tobacco smoke (cited by Pipes).[66] In 400 households, the incidence of parental smoking was about the same in asthmatic children's homes as in those of controls.[67] Speer[68] collected data on 441 nonsmokers who developed symptoms when exposed to tobacco smoke and concluded that the effect of tobacco smoke appeared to be of irritative rather than of allergic character. This he based on the following findings: similar reactions occurred in allergic and nonallergic individuals and allergic manifestations like urticaria and eczema were absent.

The question of skin testing as a form of evaluating allergy to tobacco is beyond the scope of this review because carbon monoxide is not an allergen.

c. Mental attitude. Rummel et al.[69] exposed 56 nonsmoking college students in a closed environment with cigarette smoke. Those who objected to being in the presence of cigarette smoke have significantly higher heart rates than those who did not object. In another study, Kidd[70] reported better scores in examinations for both smokers and nonsmokers when they were tested in separate rooms.

than when the two groups took the same examination together in one room. The low scores were explained by the investigator as an emotional reaction of the nonsmoker and possible guilt feelings of the smoker from the disapproval expressed by nonsmokers.[70]

E. PRACTICAL ASPECTS OF NONSMOKER QUESTION

According to the information reviewed in this chapter, the presence of cigarette smoke in public places is not a health hazard to the nonsmoker for the following reasons: First: actual measurements in public places show low levels of carbon monoxide and nicotine. Second: There is no evidence that nonsmokers, when exposed to cigarette smoke, develop cardiovascular reaction or suffer respiratory distress. The subjective complaints of smokers are a combination of the psychosocial reaction with irritation of mucosa in extreme conditions of excessive amounts of cigarette smoke in poorly-ventilated enclosures. Several authorities have concluded that cigarette smoke in public places is not a health hazard to the nonsmoker.[71-75] The opposite conclusion by some authorities[76-86] is based on the observations of elevated levels of carbon monoxide seen in chambers or in nonventilated rooms, and a misinterpretation of data on tobacco allergy and respiratory diseases of children. At the present time, the question of the effect of cigarette smoke on the nonsmoker continues to be controversial. The answer will depend ultimately on the consideration of a more fundamental question, i.e. the difference between smokers and nonsmokers in general. These two groups are apparently distinguishable by factors other than the use of cigarettes.

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